Jejunal hemorrhage syndrome in dairy and beef cattle: 11 cases (2001 to 2003)

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Abstract — The medical records of 11 cattle with jejunal hemorrhage syndrome were reviewed. Female and male, lactating and pregnant, dairy and beef cattle were affected. Decreased feed intake and milk production, reduced amounts of dark feces, and abdominal discomfort were common historical findings. Common clinical findings included depressed demeanor, a "ping" and fluid-splashing sounds over the right abdomen, melena, and distended loops of intestine on rectal palpation. Surgery was done on 7 cases, 10 cases were euthanized, and 1 died. *Clostridium perfringens* type A was isolated from the intestinal contents from 7 of 7 cases. At necropsy, the characteristic finding was a varying length of a dark purple-red distended jejunum with an intraluminal blood clot. Histologically, there was segmental necrosis, ulceration, and mucosal and transmural hemorrhage of the jejunum. This is a sporadic disease of adult cattle characterized by mechanical obstruction of the small intestines by a large blood clot with a case fatality of almost 100%.

Résumé — Syndrome de l'hémorragie jéjunale chez les bovins laitiers et de boucherie : 11 cas (2001–2003). Les dossiers médicaux de 11 bovins présentant le syndrome de l'hémorragie jéjunale ont été revus. Femelles et mâles, en lactation ou en gestation, laitiers ou de boucherie étaient affectés. Diminution de l'ingestion et de la production laitière, réduction de la quantité de fèces foncées et inconfort abdominal constituaient l'essentiel de l'historique. Les trouvailles cliniques les plus fréquentes comprenaient une attitude déprimée, un «ping» et un bruit de clapotement au niveau de l'abdomen droit, du méléna et une distension des anses intestinales à la palpation rectale. Une chirurgie a été effectuée sur 7 animaux, 10 ont été euthanasiés et 1 est mort. Du *Clostridium perfringens* type A a été isolé du contenu intestinal de 7 cas sur 7. À la nécropsie, la trouvaille caractéristique était une longueur variable de jéjunum distendu d'une couleur pourpre-rouge foncée avec caillot sanguin intraluminal. À l'histologie, on retrouvait une nécrose segmentaire, une ulcération et une hémorragie de la muqueuse et de toute l'épaisseur de la paroi du jéjunum. Il s'agit d'une maladie sporadique des bovins adultes caractérisée par une obstruction mécanique du grêle par un gros caillot sanguin accompagné d'un taux de mortalité de près de 100 %.

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Introduction

Jejunal hemorrhage syndrome (JHS) has been reported in both beef and dairy cows (1–6). The disease occurs sporadically with a case fatality rate ranging from 85% to 100% (5). The syndrome has been reported in Canada, the United States, and Germany (2–7). Mechanical obstruction of varying lengths of jejunum with sloughed mucosa and clotted blood is characteristic (1,2). The etiology is unknown. *Clostridium perfringens* type A has been suggested as the cause, because the organism has been isolated from the lesions and feed samples of clinical cases (5). However, experimental inoculation of a pure culture of *C. perfringens* type A, isolated from clinical cases, into the proximal jejunum of 12 adult nonlactating dairy cows failed to produce the syndrome (1). A history of sudden death, or of decreased milk production, anorexia, abdominal discomfort, depression, abdominal distention, melena, and distended loops of intestines on rectal examination has been reported (3,4). Neither surgical nor medical treatment has been totally successful, and the prognosis for affected cows is very unfavorable (1,4).

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Materials and methods

Criteria for selection of cases

The medical records of cattle diagnosed with JHS in the Large Animal Clinic of the Western College of Veterinary Medicine, between January 1, 2001, and October 31, 2003, were reviewed. All medical records of cattle coded under jejunitis were reviewed; those that had enteritis

Table 1. The signalment and vaccination history of 11 cattle with jejunal hemorrhage syndrome

	Age (years)	Breed	Sex	Herd size	Stage of production (days post calving)	Vaccination history
1	3	Holstein Friesian	F	80	60	Not available
2	4	Holstein Friesian	F	100	30	Not vaccinated
3	1.5	Holstein Friesian	F	65	Pregnant heifer	Not vaccinated
4	4	Holstein Friesian	F	124	75	Not vaccinated
5	3	Holstein Friesian	F	124	90	Not vaccinated
6	5	Holstein Friesian	F	140	30	Not vaccinated
7	3	Holstein Friesian	F	87	105	Not vaccinated
8	3	Holstein Friesian	F	200	Not available	Not vaccinated
9	5	Simmental	F	120	Not available	*8-way clostridia[a] *bovine viral diarrhea virus (type 1 and 2)[b] **Campylobacter fetus* vaccines[c]
10	3	Simmental	F	140	45	*7-way *clostridia* and *haemophilus somnus*[d] *bovine respiratory syncytial virus, parainfluenza-3 virus and infectious bovine rhinotracheitis virus vaccines[e]
11	3	Charolais	M	15	Not applicable	Not vaccinated

[a]Ultrabac 8, Pfizer Canada, London, Ontario
[b]Triangle 1 + Type II BVD, Ayerst Canada, Guelph, Ontario
[c]Vibrin, Pfizer Canada, London, Ontario
[d]Ultrabac 7/Somubac, Pfizer Canada, London, Ontario
[e]Bovi-Shield, Pfizer Canada, London, Ontario

and an obstructive blood clot in the jejunum, detected by exploratory laparotomy, at necropsy, or both, without evidence of an intestinal lesion that might cause primary large or small intestinal obstruction (extra luminal mass, volvulus, foreign body, intussusception), were included.

Methods of analysis

The admission date, history, source (farm), age, breed, sex, duration of clinical signs, reproductive status (females), clinical findings, results of laboratories diagnostic tests, treatment and response to treatment, microbiological testing, and necropsy findings were recorded.

A descriptive statistical analysis was done using a computerized statistical package (Student Statistix 7 Analytical Software, Tallahassee Florida, USA).

Results

Eleven adult cattle (Table 1) met the criteria for this study. Nine of the 11 cases were from different herds. The disease occurred most commonly (n = 7 cases) between December and May, with 3 cases occurring in February. Four cases were presented in 2001, 5 in 2002, and 2 from January 1 to October 1, 2003. The cases included both dairy (n = 8) and beef (n = 3) cattle. The vaccination history was available for 9 cases (Table 1). The feed management was available for 11 cases and is summarized in Table 2.

Affected cases were Holstein (n = 8), Simmental (n = 2), and Charolais (n = 1). Both sexes were affected: females (n = 10) and male (n = 1). The age of the affected cases ranged from 1.5 to 5 y (mean 3.4, s = 1 y). One case was a pregnant heifer; 7 female cases were between 1 and 3.5 mo postpartum (mean 2.1, s = 1 mo); the information was not available for 2 cases. The duration of clinical signs at admission (n = 10 cases) ranged from 0.5 to 6 d (mean 1.85, s = 1.8 d). The rectal temperatures (n = 9) ranged from 33°C to 38.3°C (mean 36.9, s = 1.7°C). The heart rates ranged from 68 to 170 beats/min (mean 105.4, s = 29.7 beats/min). The respiratory rates (n = 9) ranged from 8 to 44 breaths/min (mean 28.9, s = 13.7 breaths/min).

Findings on rectal examination were available for 8 cases, 6 of which had distended and tense loops of intestines and 2 had a distended rumen. Other clinical signs are summarized in Table 3. Blood gas analysis and serum electrolyte measurements were available for 8 cases (Table 4). Commonly, findings were metabolic alkalosis with compensatory respiratory acidosis (n = 6), hypochloremia (n = 6), and hypokalemia (n = 8). Hematologic analyses were done in 4 cases (Table 5): The main findings were leukocytosis and mature neutrophilia (n = 2), increased band neutrophils (n = 4), and fibrinogen (n = 1). Intravenous fluid therapy was given to 6 cases and consisted of different amounts of normal saline or Ringer's solution, with or without potassium chloride (20 mEq/L), according to the needs of the case. Five cases received the antibiotic trimethoprim and sulfadoxine combination (Trivetrin; Schering-Plough Animal Health, Pointe-Clair, Quebec), 16 mg/kg body weight (BW), IV, q24h, or oxytetracycline hydrochloride (Tetraject LP; Bimeda-MTC Animal Health, Cambridge, Ontario), 10 mg/kg BW, IV, q24h. Exploratory laparotomy was done on 7 cases; in 2 of these, the affected intestines had ruptured during manipulation. Neither medical nor surgical treatment was beneficial; 1 case died and 10 were euthanized.

Table 2. Feed management of 11 cattle affected with jejunal hemorrhage syndrome

Cattle	Feed management
Dairy cattle (n = 8)	(7/8) Barley silage Alfalfa or broom or chopped hay 9 to 12 kg of grain (dairy ration or barley)
	(1/8) Pregnant heifer on pasture (No grain)
Beef cattle (n = 3)	(1/3) Oat hay and no grain
	(1/3) Legume hay and 1 to 1.5 kg of oat daily
	(1/3) Marsh hay and gallon of oats every other day

Table 3. Clinical features of cattle with jejunal hemorrhage syndrome

Variable	Frequency	Percentage
Decreased feed intake	9/9	100
Depression	10/11	90
Decreased milk production	6/7	86
Tachycardia	9/11	82
Dehydration	6/8	75
Scant or reduced feces	6/8	75
Melena	8/11	73
"Ping" sound over right middle abdomen	7/11	64
Pale mucus membranes	5/8	63
Hypomotile rumen	5/8	63
Fluid splashing sounds on ballottement of right abdomen	6/11	55
Abdominal distention, lower half in both sides, more pronounced on the right	6/11	55
Abdominal discomfort and pain	4/11	37
Loose feces (liquid)	2/8	25
Muscle fasciculation	2/11	18
Recumbency	2/11	18
Feces with frank blood clots	1/8	13

Necropsies were done on 8 of the 11 cases. The common gross pathologic findings were variable lengths of dark purple-red distended jejunum with an intraluminal blood clot (Figure 1), an intramural hematoma (Figure 2), or ulceration. The hematomas were covered with a necrotic mucosa, which was often sloughed; in some cases, there were fibrin strands on the serosal surface of the jejunum. Also, in some cases, the small intestines proximal to the lesion were distended with fluid and ingesta, because the outflow was obstructed, and the large intestine contained dark-bloody fluid.

The most common histologic findings were multifocal submucosal edema and neutrophil infiltration, segmental necrosis, ulceration, and mucosal and transmural hemorrhage (hematoma) of the jejunum. In some cases, there was neutrophil infiltration. Frequently, the epithelium was completely sloughed, and in the area of attachment of the blood clot, the mucosa was absent (peeled off) (Figure 3).

Results from the culture of the intestinal contents from 8 cases for *Salmonella* spp. were negative. No bovine viral diarrhea virus (n = 3) or bovine coronavirus (n = 2) antigens were detected with the avidin biotin complex peroxidase method.

The contents of the affected intestines were cultured for *Clostridium* spp. in 7 cases; in all 7, *C. perfringens* type A was isolated. Alpha toxin alone was identified in 3 out of 7 cases, and both a and b toxin were identified in 1 out of 7 cases.

Table 4. Blood gas analysis values (venous blood sample) and serum electrolytes in 8 cattle with jejunal hemorrhage syndrome

	Range	Mean, s	Reference range (8)
pH	7.3 to 7.6	7.5, 0.1	7.35 to 7.50
PCO_2 (mmHg)	43 to 72	59.1, 12.1	34 to 45
HCO_3 (mmol/L)	21 to 54	40.5, 11.5	20 to 30
Na (mmol/L)	125.3 to 141.7	135.7, 5.4	132 to 152
K (mmol/L)	1.6 to 3.3	2.5, 0.5	3.9 to 5.8
Cl (mmol/L)	70 to 100	84, 11.5	95 to 110

Discussion

In the only previously reported retrospective study, the clinical picture of JHS was described in 22 lactating dairy cows from only 1 farm (4), whereas, in this study, the syndrome is described in lactating cows, a pregnant heifer, and an adult male; these were beef and dairy cattle from different farms, except for 2 dairy cows that were from the same herd at different times. The syndrome has not been previously reported in males or in pregnant heifers. This study represents the cattle population in western Canada, mainly Saskatchewan, where the number of cases has been relatively small, because it is rather a rare disease, making a retrospective study an appropriate way in which to study the disease.

Jejunal hemorrhage syndrome was first reported in 1997; the 1st case in our clinic occurred in 2001. The syndrome has also been called "acute hemorrhagic enteritis of the small intestine," "intraluminal-intramural hemorrhage of the small intestine," and "hemorrhagic bowel syndrome," invariably localized to the jejunum (3,4). Most of the cases presented to our hospital have occurred between December and May, with the peak in February. These months, especially February, are associated with very cold weather. The high stress level associated with very cold weather could predispose animals to the disease. The time since parturition, in this study, was less than previously reported cases (range, 9 to 319 d; mean, 107.5 d) (4). The mean age of the affected animals was similar to that in the previous study (mean 4 y). The oldest affected case in this study was 5 y, while it was 8 y in the previous study. The vital signs (temperature, heart rate, and respiration rate) were variable. The temperature ranged from normal to decreased. Decreased temperature was suspected to be due to dehydration, hypovolemia, and hemorrhagic anemia. The heart rate was normal in some cases and elevated in others, possibly due to anemia, hemorrhage, dehydration, and pain. Respiratory rate was decreased in some affected cases, possibly due to hypothermia or dehydration.

Unlike the previous study (4), where anorexia, decreased milk production, scant or reduced feces, and signs of abdominal pain were seen in 10%, 38%, 24%, and 4.5% of the cases, respectively, in our study, they were seen in 100%, 85.7%, 75%, and 36.3% of cases, respectively. The reason for that is unknown, but it is probably related to the severity and duration of sickness. The most common historical and clinical findings in our study were decreased feed intake, depressed demeanor, decreased milk production, tachycardia, dehydration, scant feces, and melena. In the previous retrospective study, depression, dehydration, and tachycardia were

Table 5. Hemograms in cattle with jejunal hemorrhage syndrome

	Range	Mean, *s*	Reference range (8)
Hematocrit (L/L) ($n = 8$)	0.16 to 0. 28	0.24, 0.04	0.24 to 0.46
WBC ($\times 10^9$/L) ($n = 4$)	9.2 to 34.1	16.9, 11.6	4. 0 to 12.0
Neutrophils (mature) ($\times 10^9$/L) ($n = 4$)	3.9 to 15.4	9.3, 4.9	0.6 to 4.0
Neutrophils (band cells) ($\times 10^9$/L) ($n = 4$)	0.64 to 1.08	0.9, 0.2	0 to 0.1
Fibrinogen (g/L) ($n = 4$)	4 to 9	5.75, 2.4	2 to 7

s — standard deviation; WBC — white blood cells

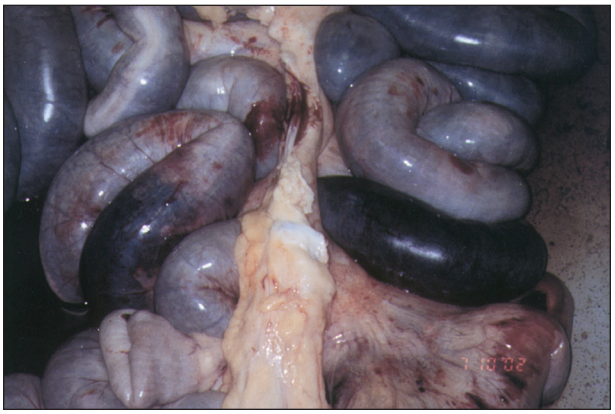

Figure 1. Dark purple-red distended small intestines with intraluminal blood clot in a dairy cow with jejunal hemorrhage syndrome.

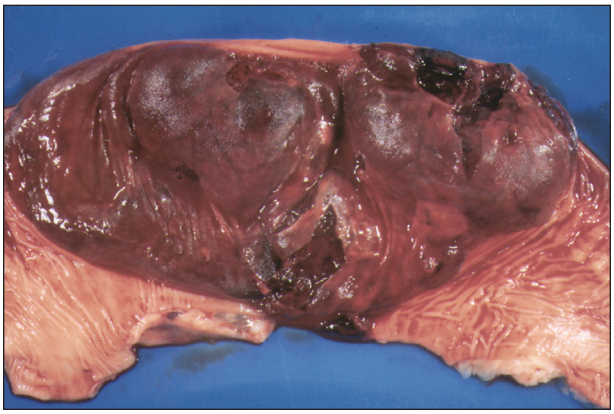

Figure 2. Intraluminal hematoma in the jejunum of a dairy cow with jejunal hemorrhage syndrome.

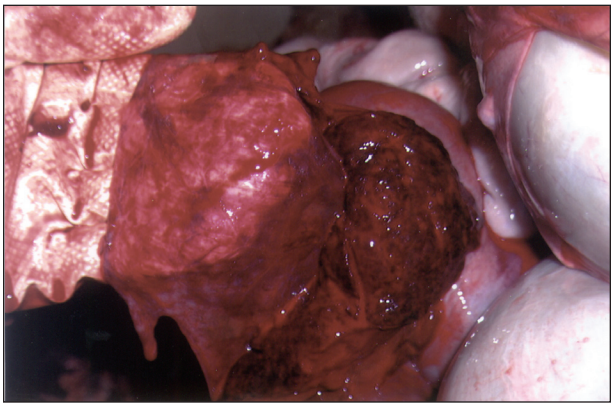

Figure 3. Sloughed jejunal mucosa (peeled off) in a bull with jejunal hemorrhage syndrome.

observed most commonly. Leukocytosis, mature and band neutrophilia, and hyperfibrinogemia were more common in the previous study (4). The range and the mean of the packed cell volume in our study (range, 0.16 to 0.28 L/L; mean 0.24, $s = 0.04$ L/L) were less than in the previous study (range, 0.29 to 0.53 L/L; mean, 0.40 L/L). Perhaps the degree of hemorrhage was more severe and the duration of clinical signs was longer in our cases.

The common findings of the venous blood gas analysis and serum electrolytes levels were metabolic alkalosis with compensatory respiratory acidosis, hypokalemia, and hypochloremia, which are consistent with abomasal outflow obstruction, caused by the clotted blood or ileus. Hypokalemia could also have been caused by decreased feed intake.

The risk factors for this syndrome include increased milk production, increased intake of soluble carbohydrates, increased energy in the diet, and decreased fiber in the diet (4,6). However, these risk factors were based on cases in lactating dairy herds and did not include beef, pregnant, or male cattle.

The etiology and pathogenesis of the syndrome are unknown. There are 2 hypotheses that assume that it is caused by *Clostridium* spp. (3). The first is that it is similar to hemorrhagic enteritis caused by *C. perfringens* type C in rapidly growing suckling calves, lambs, or piglets. The bacteria multiply rapidly and produce toxins under conditions of high carbohydrate and protein substrate availability (3). This sequence of events could possibly occur in adult dairy cows in association with factors similar to those leading to ruminal acidosis (feeding of excess amounts of fermentable carbohydrates; insufficient effective fiber, inadequate fiber mat, or both; or ration sorting by cows) (3).

The second hypothesis suggests that improper fermentation of the ensiled feeds (poor silo or bunker management) may allow the accumulation of harmful molds, clostridia, or other harmful bacteria, as well as their potential toxins, which are then ingested by the cow (3).

It is difficult to verify these hypotheses on the basis of our study, because some affected animals were on pasture and others were fed hay. Some cases were not fed grain and others were fed a relatively small amount. On the contrary, some animals were fed rations containing high carbohydrates and ensiled feed.

Clostridium perfringens Type A has been suggested as a cause of the syndrome and, in our study, it was isolated from the intestinal contents of all tested cases (7/7). The failure of inoculation of this organism into the abomasum ($n = 6$) and the proximal jejunum ($n = 6$) of adult nonlactating cows to produce the syndrome in a previous study (1), does not exclude *C. perfringens* as a causative agent.

Clostridium perfringens is a spore-forming, anaerobic, bacillus, which is believed to cause several enterotoxemic diseases in animals and humans (9). It is normally present in large numbers in feces, soil, sewage, and the intestinal tract of animals and humans (9,10). The manifestation and pathology of enterotoxemias caused by *C. perfringens* differ according to the toxigenic type and the particular toxins produced (9,10). The organism can be divided into 5 types (A, B, C, D, and E), based on the production of 4 major lethal toxins: alpha, beta, epsilon, and iota (9). Other toxins or antigenic substances, like enterotoxin, kappa (collagenase), mu (hyaluronidase), 2 hemolysins, delta, and theta, may be produced. *Clostridium perfringens* type A can produce several different toxins (alpha, beta, and enterotoxin) (9–11). Alpha toxin is the principal lethal toxin (a phospholipase C) (10). It is also hemolytic and necrotizing, and has the ability to split lecithin or lecithin-protein complexes (10). Beta toxin is lethal and responsible for inflammation of the intestine and partial loss of the mucosa (10). Enterotoxin is a single polypeptide with a unique amino acid sequence (9). It acts rapidly on the intestinal mucosa, mainly in the jejunum and ileum, causing profuse diarrhea, along with associated clinical signs (9). *Clostridium perfringens* type A has been implicated in several diseases in different animal species (8,10–15), but its role is uncertain, because it is part of the bacterial flora of alimentary tract in many normal animals (8,13), leading to considerable controversy in the literature about whether or not *C. perfringens* Type A can cause diseases like enteritis and enterotoxemia (16,17). It is associated with equine intestinal clostridiosis, diarrhea in pigs, fatal hemolytic diseases in cattle and sheep (yellow lamb disease), hemorrhagic enteritis in adult cattle and calves, and hemolytic enterotoxemia in foals (8,10–15). The key to the presumptive diagnosis of *C. perfringens* type A diseases is the presence of large numbers of the bacteria and the toxin (8,13,18,19). Extensive necrosis of the small intestine is one of the lesions in some of the previously mentioned diseases (8).

Surgical and medical therapies were unsuccessful for JHS in the cases in our study, in which all animals died or were euthanized. There are several reasons for the poor outcome of the treatment trials. These include the severity of the lesion and the financial constraints that prevented doing certain surgical procedures and long-term aftercare. In a previous study, 7 out of 8 cows treated medically died and 9 out of 13 cows treated surgically died or were euthanized (4). In another study (EM Santaschi et al, personal communication), 7 of 13 cows treated surgically recovered. The surgical procedures, which were performed on the discharged cows, were intestinal resection (1/3) (number survived/number of times procedure performed), enterotomy (2/6), and massage (4/4). The difference in the outcome of the surgical and medical treatment of the disease might be related to the stage of the disease at which the treatment was attempted and the use of a different surgical approach in dealing with the lesion.

There is no known strategy to prevent or control the syndrome. More critical investigations to identify the risk factors and ways in which to avoid them would help in reducing the prevalence of the syndrome. If *C. perfringens* Type A is the causative agent, a study to determine if a vaccine would reduce the prevalence in the farms where multiple cases are being reported may be indicated.

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